Respiratory Insufficiency in Combat Casualties:

I. Pathologic Changes in the Lungs of Patients Dying of Wounds

ARTHUR M. MARTIN, JR.,* CAPT., MC, RICHARD L. SIMMONS,** CAPT., MC, CHARLES A. HEISTERKAMP, III, LT. COL., MC

From the Division of Surgery, Walter Reed Army Institute of Research, Walter Reed Army Medical Center, Washington, D. C. 20012

PHYSICIANS actively caring for combat casualties in Vietnam have been impressed with the frequency and severity of respiratory insufficiency in the wounded. This problem occupied significant portions of two conferences on war surgery,[11, 31] and a symposium on pathogenesis was recently held under the auspices of the National Research Council-National Academy of Sciences.[30] In these discussions, it was apparent that the syndrome encompasses a variety of pulmonary complications in patients with thoracic injuries and in those without direct pulmonary trauma. These disorders have become known as the "wet lung" syndrome although Burford's original description of a syndrome by that name referred to excessive retained secretions following thoracic trauma.[5]

The present pathological and pathophysiological studies were designed to assess the extent and type of pulmonary disease in battle casualties in Vietnam. The initial paper documents the prevalence and severity of changes in the lungs of American soldiers who were resuscitated following wounding, but subsequently died. In this way the role played by respiratory insufficiency in death might be defined.

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* Present address: Department of Pathology, Duke University, Durham, North Carolina.

** Present address: Department of Surgery, University of Minnesota, Minneapolis, Minnesota.

Materials

The information was obtained from autopsy files of the U. S. Army 9th Medical Laboratory, Saigon, Vietnam. More than 800 autopsy reports were screened, covering the period from July 1966 to January 1968, from which 100 reports were analyzed. Selection was made on the basis of the following criteria: 1. All patients had received combat wounds. 2. None had been burned. 3. All were evacuated to a hospital and only four were dead on arrival. All others had received resuscitative care, survived the initial operation, and died during the postoperative period. 4. The autopsy report was sufficiently detailed, usually with microscopic study, to permit critical analysis. Information concerning the clinical courses was obtained from clinical summaries of autopsy reports. These reports, therefore, comprise all those available of patients wounded in action who subsequently died after initial therapy. Since autopsies are not routinely performed on all combat casualties in Vietnam, these reports probably were of special interest to attending physicians. Correlations between pathologic findings were determined by chi square analysis of data.

Results

The patients were young men (18–47 years mean 24.5 ± 6.8 years) and 83 of the 100 were Caucasian. Although there was a

high incidence of injuries to the abdomen and extremities (Table 1), the distribution of major sites of injury was reasonably uniform (Table 2). The major site in patients with multiple wounds was that determined by the prosector to be the most significant which contributed to the patient's death. There were 74 major operations (craniotomy, open thoracotomy, laparotomy, and amputations); the remainder of operations were wound debridements. Twenty-five of the 100 patients required transfusions of more than 20 units of whole blood.

Mean survival time following injury was 6.5 days (Table 2). Four were dead on arrival at the hospital. Most deaths occurred during the first week (62%) with the largest number (33%) within the first 24 hours (Fig. 1). Patients with head or chest injuries did not live as long as the average for the series (Table 2); only two patients with these injuries lived longer than 7 days.

The lung weight was considered the single best objective indicator of pulmonary disease. Mean combined lung weight was 1,783 grams. Of recorded lung weights, 64.6% were between 1,000 Gm. and 2,000 Gm., and 30.4% were in excess of 2,000 grams. Only 5.0% weighed less than 1,000

TABLE 1. *Incidence of Injuries*

Head	21		
Blunt		14	
Penetrating		7	
Skull fracture		17	
Chest	32		
Penetrating		22	
Blunt		10	
Abdomen	41		
Colon		19	
Small intestine		18	
Liver		14	
Spleen		13	
Kidney		7	
Urinary bladder		5	
Stomach		3	
Pancreas		1	
Extremity & soft tissue	49		
Extremity		44	
Upper			25
Lower			52
Neck		5	
Fractures	114		
Tibia		18	
Femur		13	
Humerus		8	
Other (rib, vertebra, etc)		75	
Arterial	18		

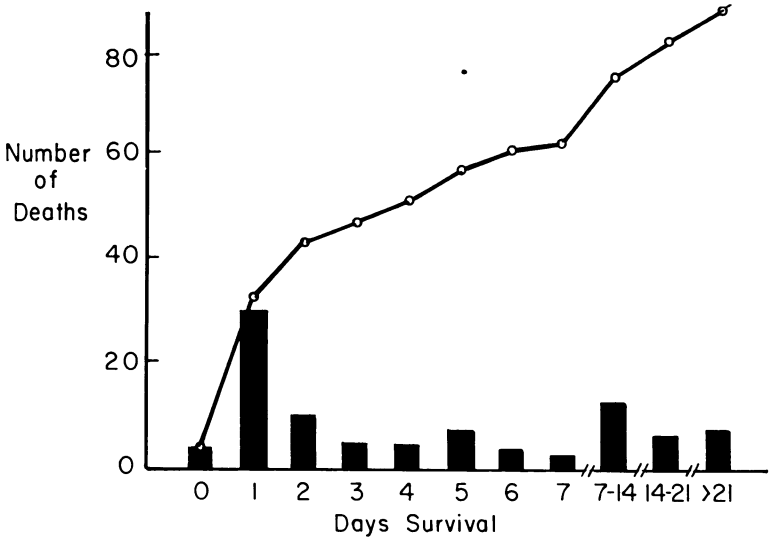

FIG. 1. Relationship between the number of deaths and the duration of survival in 89 patients in whom the length of survival is precisely known. The bar graph represents the number of deaths at each time interval, the cumulative number of deaths is indicated by the ascending line.

TABLE 2. *Relationship of Site of Major Injury to Duration of Survival*

Duration of Survival (Days)	All Cases	Head*	Chest*	Abdomen*	Thoraco-abdominal*	Extremity Only	Major Injury not Specified
Mean	6.5	3.8	4.1	7.9	8.8	9.0	5.4
Standard deviation	±5.66	±7.03	±5.03	+9.33	±8.70	±9.76	±6.64
No. of cases	89†	17	13	22	13	12	12

* Includes those cases having associated extremity and soft tissue injuries.
† Includes those cases in which the period of survival was not recorded.

grams. There was no correlation between lung weights and duration of illness or site of injury. For deaths during the first week following either head injuries or abdominal injuries, there was a slight correlation between length of survival and lung weight (Fig. 2).

Table 3 lists the incidence of findings by organ systems. Respiratory diseases were the most frequent at autopsy in all patients whether or not thoracic trauma had been sustained. Only hemothorax and gross atelectasis were significantly more common following thoracic injury (Table 4).

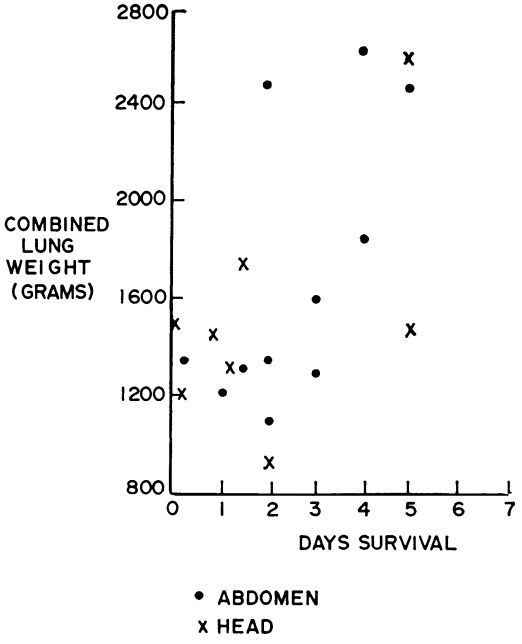

FIG. 2. Relationship of combined lung weight to duration of survival for patients with head or abdominal wounds.

Pulmonary edema, congestion and alveolar hemorrhage were found in 89 of the 100. These findings were more common in patients dying within the first week following injury (Fig. 3 and Table 5). Congestion, edema, and hemorrhage were not often complicated by pneumonia or hyaline membrane disease during the first few days after injury. Pleural effusion was a frequent concomitant finding to pulmonary edema (Table 6).

Bronchopneumonia was present in 29 patients. There was a lesser incidence in those with head injuries. The incidence of pneumonia increased as the interval from injury to death increased (Table 5). Pneumonia was more frequent in lungs weighing more than 2,000 Gm. ($p < 0.05$) (Table 7).

Similarly, microscopic hyaline membranes were described in 11 of the 100 reports. The membranes were characterized by thick eosinophilic material covering alveolar walls, respiratory ducts, and terminal bronchioles. The period of survival was longer (mean of 10.4 days) than with other findings. All but two of these patients survived at least 5 days. The lungs in these instances were significantly heavier than the rest ($p < 0.01$) (Table 7).

Thromboemboli were reported in the lungs of 13 subjects. In eight of these pulmonary infarction developed. There was no relationship between the presence of em-

TABLE 3. *Pathologic Anatomy of 100 Cases of Combat Deaths*

Cardiovascular
- 12 Right ventricular dilatation
- 16 Subendocardial hemorrhage
- 10 Left ventricular dilatation
- 4 Cardiac hypertrophy
- 4 Penetrating wound of the heart
- 4 Myocardial infarction
- 2 Myocardial abscess

Heart weight, mean, 356 g.

Respiratory
- 73 Pulmonary edema
- 50 Pulmonary congestion
- 48 Pleural effusion, mean, 677 ml
- 43 Tracheostomy
- 32 Chest injury
  - 22 Penetrating
  - 10 Blunt
- 29 Bronchopneumonia
  - 4 Abscess
- 28 Pulmonary hemorrhage
- 16 Hemothorax, mean, 703 ml
- 13 Pulmonary thromboemboli
  - 8 Pulmonary infarction
- 11 Pulmonary hyaline membrane
- 11 Atelectasis
- 4 Pulmonary fat embolism

Lung weight, combined mean 1783 g.

Gastrointestinal
- 41 Abdominal injury
- 19 Colon
- 18 Small intestine
- 14 Liver
- 3 Stomach
- 1 Pancreas
- 27 Hepatic congestion
- 24 Retroperitoneal hemorrhage, mean 1,168 ml
- 13 Gastric ulcer
- 12 Ascites, mean, 470 ml
- 10 Peritonitis
- 7 Hepatic fatty metamorphosis
- 6 Hepatic necrosis
- 6 Gastrointestinal hemorrhage
- 6 Hemoperitoneum, mean, 920 ml
- 1 Hemorrhagic gastritis
- 1 Pancreatitis

Liver weight, mean, 2002 g.

Genitourinary
- 18 Acute tubular necrosis
- 12 Injury
  - 7 Kidney
  - 3 Urinary bladder
- 10 Hemorrhagic cystitis
- 10 Nephrectomy
- 7 Renal fat embolism
- 4 Pyelonephritis
- 1 Glomerulitis

Kidney weight, combined mean 414 g.

Endocrine
- 5 Adrenal lipid depletion
- 2 Adrenal atrophy
- 2 Adrenal hemorrhage

Hematopoietic
- 14 Splenic congestion
- 13 Splenic injury
  - 11 Splenectomy
- 2 Acute splenitis

Spleen weight, mean, 274 g.

Central Nervous System
- 21 Head injury
  - 14 Blunt
  - 7 Penetrating
  - 17 Skull fracture
- 19 Cerebral edema
- 18 Subarachnoid hemorrhage
- 14 Subdural hemorrhage
- 14 Intracerebral hemorrhage
- 11 Cerebral contusion
- 8 Craniotomy
  - 3 Craniectomy
- 8 Cerebral fat embolism
- 3 Cerebral infarction

Brain weight, mean, 1507 g.

Musculoskeletal
- 114 Fractures
  - 18 Tibia
  - 13 Femur
  - 8 Humerus
  - 75 Other (rib, vertebra, etc)
- 44 Extremity injury
  - 52 Lower
  - 25 Upper
- 18 Arterial injury
- 14 Amputations
  - 7 Below knee
  - 5 Above knee
  - 1 Knee disarticulation
  - 1 Above elbow
  - 1 Below elbow
- 5 Penetrating neck injury

TABLE 4. *Type of Injury vs Pulmonary Diagnosis*

Diagnoses	Thoracic	Nonthoracic
Bronchopneumonia	9	15
Edema, congestion and/or hemorrhage	29	48
Pleural effusion	14	28
Hyaline membranes	4	4
Hemothorax	11*	5
Atelectasis	7†	4
Number of cases	32	54

* Incidence significantly greater ($p < 0.01$).
† Incidence significantly greater ($p < 0.05$).
Note: In 14 cases the site of injury was not specified.

boli and either the site of major injury or other pathologic findings.

Fat embolism of the lungs was recorded in only four of the 100 reports. Each of these patients had sustained extremity injuries, three lower and one upper, with fractures of the femur, tibia and humerus. Three patients died within 24 hours and the other survived for 2 weeks. In each case there was systemic fat embolization; the brain was involved in all four, and the kidney in three. In seven additional cases systemic fat embolism was present (three in the kidney, three in the brain, and one in both) but no mention was made as to whether or not the lungs were involved. In every instance of fat embolism, whether pulmonary or systemic, pulmonary edema was present ($p < 0.05$). Undoubtedly, there were other cases of fat embolism; the low incidence is most likely related to the fact that examination for fat was not routinely performed.

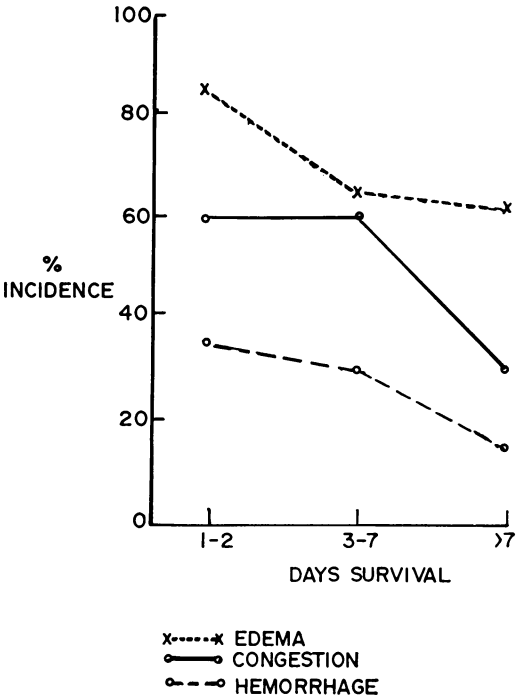

FIG. 3. Relationships of the incidences of pulmonary edema, congestion and hemorrhage to the length of survival.

Serious systemic infections and renal failure, are thought to be causally related to severe respiratory insufficiency.[6, 25] Septicemia developed in 18 patients (Tables 5, 6). A gram negative bacillus (*E. coli*, Klebsiella-Aerobacter, Pseudomonas, or Proteus) was the usual organism identified. Most septicemias occurred following abdominal injury ($p < 0.01$), and developed after the first week of survival ($p < 0.001$). Bronchopneumonia was most frequently seen in these subjects ($p < 0.05$). There were 18 instances of renal tubular necrosis. The incidence was greater after the first week. Thirteen followed abdominal injury ($p < 0.01$). There was a correlation between the incidence of renal tubular necrosis and pulmonary hyaline membranes ($p < 0.05$) and bronchopneumonia ($p < 0.05$) (Table 6).

A diagnosis of acute congestive failure was frequently made by the prosector in these patients because of extensive pulmonary edema even in the absence of significant cardiac pathologic changes. Myocardial changes found are listed in Table 3. All patients with myocardial infarcts, myocardial abscesses or penetrating wounds except one survived more than one week. The estimated age of myocardial infarcts corresponded to the interval between wounding and death. The oldest patient in this group was 30 years of age.

TABLE 5. *Relationship of Pathologic Findings to Duration of Survival*

Duration of Survival	Total Deaths	Pulmonary Hemorrhage	Pulmonary Emboli	Pulmonary Congestion	Hemothorax	Pulmonary Edema	Pleural Effusion	Hyaline Membrane	Bronchopneumonia	Sepsis	Renal Tubular Necrosis
<7 days	63	21	10	36*	14	48	27	7	15	6	8
>7 days	26	4	2	8	2	16	10	4	14†	12ˣ	8
Total	89	25	12	44	16	64	37	11	29	18	16

* Significant incidence $p < 0.05$.
† Significant incidence $p < 0.01$.
ˣ Significant incidence $p < 0.001$.

Discussion

In the Vietnam conflict, evacuation from the field is rapid, blood is plentiful, medical specialists are present at every echelon, and hemodialysis is readily available.[5] The period of hypovolemic shock has thus been shortened and the contribution of renal failure to death has been lessened. But the autopsy incidence of severe pulmonary disease in combat casualties has not changed in 25 years.[25] Pulmonary edema, congestion, alveolar hemorrhage, atelectasis and pneumonia were frequent in the lungs of soldiers who died of wounds in World War II.[25] Previous emphasis, however, was on the role of fat embolism, renal failure and prolonged untreated shock in the mechanism of death.[25]

The present study was designed to determine the prevalence of pulmonary disease in combat casualties who survive initial resuscitation but subsequently die. The deficiencies of this type of retrospective study are obvious: Clinical correlation is difficult, pathological descriptions are frequently incomplete, organ weights are not recorded in all reports, pulmonary fat embolism is

TABLE 6. *Cross Correlation of Pathologic Diagnoses*

Pulmonary Diagnosis	Number of Patients with Diagnosis Listed								
	Total Number	Pulmonary Edema Congestion Alveolar Hemorrhage	Pneumonia	Pleural Effusion	Hemothorax	Pulmonary Emboli	Gross Atelectasis	Hyaline Membranes	Renal Tubular Necrosis
Pulmonary edema									
Congestion									
Alveolar hemorrhage	89								
Pneumonia	29	26							
Pleural effusion	47	41	16						
Hemothorax	16	13	3	3					
Pulmonary emboli	13	11	6	4	3				
Gross atelectasis	11	8	4	6	4	3			
Hyaline membranes	11	9	8†	7	1	2	4**		
Renal Tubular necrosis	18	16	9*	10	2	4	2	5	
Sepsis	18	15	9*	11	1	2	4	3	7*

* Significant correlation $p < 0.05$.
** Significant correlation $p < .001$.
† Significant correlation $p < 0.001$.

TABLE 7. *Relationship of Pathologic Findings to Lung Weights*

Lung Weights (Gm.)	No. of Cases	Bronchopneumonia	Pulmonary Hemorrhage	Pulmonary Emboli	Pulmonary Congestion	Pulmonary Edema	Pleural Effusion	Hyaline Membrane	Hemothorax	Renal Tubular Necrosis	Sepsis
<1,000	4	1	1	1	0	3	1	0	0	0	2
1,001–1,500	24	5	5	3	17	21	10	0	3	4	6
1,501–2,000	27	8	12	3	11	20	17	2	2	3	5
>2,000	24	12*	6	2	15	18	13	8x	4	5	3
Total	79	26	24	9	43	62	41	10	9	12	16

* Greater incidence of pneumonia in lungs weighing more than 2,000 Gm. ($p < 0.05$).
x Greater incidence of hyaline membranes in lungs weighing more than 2,000 Gm. ($p < 0.001$).

not routinely sought, and even routine histologic study is occasionally omitted. Despite such shortcomings, pulmonary damage was found prevalent and extensive. Lung weights best reflected this finding: 95% of patients had combined lung weights in excess of 1,000 Gm.; 64% were greater than 1,500 Gm., and 24 in excess of 2,000 grams. This weight gain is the sum of all pathological processes and is some measure of respiratory functional impairment. There were no differences between lung weights of patients with direct pulmonary trauma and those without. With the exception of hemothorax and gross atelectasis, the type of pulmonary damage was similar following thoracic and nonthoracic injury.

A pattern of pulmonary changes could be discerned. Edema vascular congestion and alveolar hemorrhage appeared most frequently in the lungs of patients dying in the immediate post-resuscitative period. These patients die with "wet lungs." Viewed from the perspective of laws governing fluid exchange across the capillary membrane,[39] Table 8 lists those factors which might foster the development of pulmonary edema following wounding and resuscitative treatment. The exact role of each factor is impossible to determine in retrospect. It is equally difficult to ascribe precise roles to myocardial damage, renal tubular necrosis, fat emboli, thromboemboli or sepsis found. The data support Moore's suggestion, however, that the lungs are a vulnerable target organ for a variety of pathogenetic stimuli.

Thus, early pulmonary edema, congestion and hemorrhage may not only lead to early respiratory insufficiency, but may also pre-

TABLE 8. *Possible Pathogenetic Factors in the Formation of Pulmonary Edema in Combat Casualties*

I. Increased pulmonary capillary permeability
   A. Direct trauma: missile; "blast"[41]
   B. Aspiration
   C. Pulmonary hypoxia
      1. Hypoperfusion[9, 16, 19, 38]; neurovascular reflexes[20, 21, 34]; hypocarbia[34]
      2. Vascular obstruction: fat and tissue emboli[7, 8]; thrombo-emboli[23]; platelet thrombi[1, 36]; disseminated intravascular coagulation[14]
   D. Toxins: fatty acids[7, 8]; histamine[37]; serotonin[37]; kinins[35]; wound endotoxins[35]; inhaled gases; lysosomes[18]; catecholamines[2]; acidosis[3, 22]; oxygen[29]
   E. Homologous blood: transfusion reactions[13]; graft-versus host reaction[12]
   F. Pulmonary infections
II. Increased pulmonary capillary pressure
   A. Neurovascular reflexes: CNS injury[10]; postcapillary arteriolar constriction[38]; postcapillary pulmonary venoconstriction[9]; systemic vasoconstriction with fluid shifts to the lesser circuit; loss of left ventricular compliance[24]
   B. Overtransfusion
   C. Myocardial failure
III. Diminished intravascular oncotic pressure: excessive crystalloid infusion; hypoproteinemia
IV. Decreased intra-alveolar pressure
V. Increased tissue oncotic pressure
VI. Surfactant deterioration[16]

pare the pulmonary bed for the development of chronic changes. In addition to pneumonia, pulmonary hyaline membranes were a frequent later finding. Hyaline membrane formation is a response to pulmonary injury by a variety of agents [4, 17, 26, 27, 33, 40] including shock and oxygen therapy.[29, 32] In experimental preparations the earliest changes are edema, congestion of the alveolar septae and disruption of the capillary endothelium. Intra-alveolar edema follows accompanied by swelling and necrosis of alveolar and bronchiolar epithelium. As the damaged epithelial cells are sloughed, hyaline membrane laminates on the denuded surfaces.[32] Thus, experimental findings are in agreement with actual findings that pulmonary hyaline membranes are related to acute congestive, hemorrhagic and edematous lesions in wounded patients. Almost all reported patients had progressive respiratory insufficiency, either from alveolar capillary block, or from hypoventilation of involved alveolar ducts. Subsequent papers will deal with respiratory functional deficits in combat casualties who survive.

Summary

1. The pathological findings in the lungs of 100 wounded soldiers who died after resuscitation are reviewed. Of the 79 reports of lung weights 95% of lungs weighed more than 1,000 Gm.; 65% more than 1,500 Gm., and 30% greater than 2,000 grams.

2. Soldiers who died within a few days of injury most frequently had combinations of pulmonary edema, alveolar hemorrhage and vascular congestion. Pleural effusion, pneumonia and pulmonary hyaline membranes occurred most frequently in patients who died later.

3. A variety of factors may contribute to the development of pulmonary edema, hemorrhage and congestion in the acutely wounded combat casualty. These changes in surviving patients may predispose to the development of pneumonia and pulmonary hyaline membranes.

4. The evidence suggests that pulmonary insufficiency may play a major role in the death of young previously healthy men wounded in Vietnam.

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